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=> d 11

L1 ANSWER 1 OF 2 REGISTRY COPYRIGHT 2009 ACS on STN

RN 6899-04-3 REGISTRY

ED Entered STN: 16 Nov 1984

CN Glutamine (CA INDEX NAME)

OTHER NAMES:

 $CN \qquad (\pm)-Glutamine$ 

CN  $\gamma$ -Glutamine

CN DL-Gln

CN DL-Glutamine

CN NSC 97925

DR 585-21-7

MF C5 H10 N2 O3

CI COM

LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN\*, BIOSIS, BIOTECHNO, CA, CAPLUS, CASREACT, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, DETHERM\*, EMBASE, GMELIN\*, IPA, MEDLINE, MRCK\*, PIRA, PROMT, SPECINFO, TOXCENTER, USPAT2, USPATFULL

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256 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
256 REFERENCES IN FILE CAPLUS (1907 TO DATE)

=> file caplus
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FILE LAST UPDATED: 24 Sep 2009 (20090924/ED)
REVISED CLASS FIELDS (/NCL) LAST RELOADED: Jun 2009
USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Jun 2009

CAplus now includes complete International Patent Classification (IPC) reclassification data for the third quarter of 2009.

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=> s 11 L2 30059 L1

=> s 12 and radiation 828052 RADIATION 14160 RADIATIONS 833881 RADIATION

(RADIATION OR RADIATIONS)

L3 278 L2 AND RADIATION

=> s 13 and normal(A)tissue

1080030 NORMAL

6132 NORMALS

1082466 NORMAL

(NORMAL OR NORMALS)

849208 TISSUE

401723 TISSUES

1077365 TISSUE

(TISSUE OR TISSUES)

21528 NORMAL(A)TISSUE

L4 5 L3 AND NORMAL(A)TISSUE

=> d 14 1-5 ibib abs

L4 ANSWER 1 OF 5 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2008:1015298 CAPLUS

DOCUMENT NUMBER: 150:120585

TITLE: Glutamine affects glutathione recycling enzymes in a

DMBA-induced breast cancer model

AUTHOR(S): Kaufmann, Yihong; Todorova, Valentina K.; Luo, Shaoke;

Klimberg, V. Suzanne

CORPORATE SOURCE: Medical Research Service, Central Arkansas Veterans

Healthcare System, Little Rock, AR, USA

SOURCE: Nutrition and Cancer (2008), 60(4), 518-525

CODEN: NUCADQ; ISSN: 0163-5581

PUBLISHER: Taylor & Francis, Inc.

DOCUMENT TYPE: Journal LANGUAGE: English

Malignancy depletes host glutathione (GSH) levels to increase treatment-related toxicity and increases itself to resist the treatments. Our previous studies have shown that dietary glutamine (GLN) prevented 7,12-dimethylbenz[a]anthracene (DMBA)-induced mammary tumors through enhancing gut GSH release and reducing tumor GSH level. In addition, GSH synthesis, metabolism, and recycling are accomplished in  $\gamma$ -glutamyl cycle. We hypothesized that the GLN prevention might be through a differential regulation of the  $\gamma$ -glutamyl cycle enzymes. Female Sprague-Dawley rats were randomized into DMBA-tumor bearing, DMBA-treated, and control groups subdivided into GLN and water groups. GLN supplementation was given at 1 g/kg/day by gastric gavage. The activities and mRNA levels of  $\gamma\text{-glutamyl}$  transpeptidase (GTP),  $\gamma$ -glutamylcysteine synthetase (GCS), 5-oxo-L-prolinase (OPase),  $\gamma$ -glutamyl transferase (GTF), and glutaminase (GLNase) were determined in gut mucosa and breast tumor using specific enzyme assays and semiquant. reverse transcription polymerase chain reaction. GLN upregulated gut GTP, GCS, OPase, and GLNase in DMBA-tumor bearing, DMBA-treated, and/or control rats; however, it downregulated these enzymes in the tumor. The paradoxical effect of GLN on key GSH recycling enzymes in the gut vs. tumor suggests that dietary supplemental GLN could be used in the clin. practice to increase the therapeutic index of cancer treatments by protecting normal tissues from, and sensitizing tumor cells to, chemotherapy and radiation-related injury.

OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (2 CITINGS)

REFERENCE COUNT: 49 THERE ARE 49 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 2 OF 5 CAPLUS COPYRIGHT 2009 ACS on STN ACCESSION NUMBER: 2005:316322 CAPLUS

DOCUMENT NUMBER: 142:367705

TITLE: Site and rate selective prodrug formulations of D609

with antioxidant and anticancer activity

INVENTOR(S): Meier, G. Patrick; Bai, Aiping; Zhou, Daohong

PATENT ASSIGNEE(S): MUSC Foundation for Research Development, USA

SOURCE: PCT Int. Appl., 86 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

	PATENT NO.				KIND		DATE		APPLICATION NO.						DATE			
					A2 A3	A2 20050414 A3 20070412		WO 2004-US33255				20041008						
	WO	W:		-				AU,		BA,	BB,	BG,	BR,	BW,	BY,	BZ,	CA,	CH,
			CN,	CO,	CR,	CU,	CZ,	DE,	DK,	DM,	DZ,	EC,	EE,	EG,	ES,	FI,	GB,	GD,
			GE,	GH,	GM,	HR,	HU,	ID,	IL,	IN,	IS,	JP,	KE,	KG,	KP,	KR,	KΖ,	LC,
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			NO,	NZ,	OM,	PG,	PH,	PL,	PT,	RO,	RU,	SC,	SD,	SE,	SG,	SK,	SL,	SY,
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		RW:	BW,	GH,	GM,	ΚE,	LS,	MW,	MZ,	NA,	SD,	SL,	SZ,	TZ,	UG,	ZM,	ZW,	ΑM,
			ΑZ,	BY,	KG,	KΖ,	MD,	RU,	ΤJ,	TM,	ΑT,	BE,	BG,	CH,	CY,	CZ,	DE,	DK,
			EE,	ES,	FΙ,	FR,	GB,	GR,	HU,	ΙE,	ΙΤ,	LU,	MC,	NL,	PL,	PT,	RO,	SE,
			SI,	SK,	TR,	BF,	ВJ,	CF,	CG,	CI,	CM,	GΑ,	GN,	GQ,	GW,	ML,	MR,	ΝE,
			SN,	TD,	TG,	ΑP,	EA,	EP,	OA									
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PRIOR	PRIORITY APPLN. INFO.:									US 2003-509700P					P 20031008			
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## OTHER SOURCE(S): MARPAT 142:367705

AB Compds. that are heteroatom substituted alkyl derivs. of tricyclodecan-9-yl-xanthogenate (D609), and pharmaceutical compns. of these compds., are disclosed. Methods of treating a disease or disorder in a subject and methods of protecting normal tissues in a subject from toxicity associated ionizing radiation or chemotherapy using compns. comprising these novel compds. are also disclosed. The invention also concerns methods of treating a disease or disorder in a subject using compns. that include these novel compds. while concurrently or consecutively treating the subject with ionizing radiation or a chemotherapeutic agent.

L4 ANSWER 3 OF 5 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2003:848354 CAPLUS

DOCUMENT NUMBER: 140:331501

TITLE: Prevention of chemotherapy and radiation

toxicity with glutamine

AUTHOR(S): Savarese, Diane M. F.; Savy, Gayle; Vahdat, Linda;

Wischmeyer, Paul E.; Corey, Barbara

CORPORATE SOURCE: Department of Medicine, Division of Hematology

Oncology, University of Massachusetts Medical School,

Worcester, MA, USA

SOURCE: Cancer Treatment Reviews (2003), 29(6), 501-513

CODEN: CTREDJ; ISSN: 0305-7372

PUBLISHER: Elsevier Science Ltd.

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review. Goals of the work: Malignancy produces a state of physiol. stress that is characterized by a relative deficiency of glutamine, a condition that is further exacerbated by the effects of cancer treatment. Glutamine deficiency may impact on normal tissue tolerance to antitumor treatment, and may lead to dose redns. and

compromised treatment outcome. Providing supplemental glutamine during cancer treatment has the potential to abrogate treatment-related toxicity. We reviewed the available data on the use of glutamine to decrease the incidence and severity of adverse effects due to chemotherapy and/or radiation in cancer patients. Methods: We performed a search of the MEDLINE database during the time period 1980-2003, and reviewed the English language literature of both human and animal studies pertaining to the use of glutamine in subjects with cancer. We also manually searched the bibliogs. of published articles for relevant refs. Main results: The available evidence suggests that glutamine supplementation may decrease the incidence and/or severity of chemotherapy-associated mucositis, irinotecan-associated diarrhea, paclitaxel-induced neuropathy, hepatic veno-occlusive disease in the setting of high dose chemotherapy and stem cell transplantation, and the cardiotoxicity that accompanies anthracycline use. Oral glutamine supplementation may enhance the therapeutic index by protecting normal tissues from, and sensitizing tumor cells to chemotherapy and radiation -related injury. Conclusions: The role of glutamine in the prevention of chemotherapy and radiation-induced toxicity is evolving. Glutamine supplementation is inexpensive and it may reduce the incidence of gastrointestinal, neurol., and possibly cardiac complications of cancer therapy. Further studies, particularly placebo-controlled phase III trials, are needed to define its role in chemotherapy-induced toxicity.

OS.CITING REF COUNT: 47 THERE ARE 47 CAPLUS RECORDS THAT CITE THIS

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FORMAT

L4 ANSWER 4 OF 5 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 1996:566715 CAPLUS

DOCUMENT NUMBER: 125:296227

ORIGINAL REFERENCE NO.: 125:55311a,55314a

TITLE: Effects of the amino acid glutamine on frequency of

chromosomal aberrations induced by gamma

radiation in Wistar rats

AUTHOR(S): Crispim Tavares, Denise; Takahashi, Catarina S.

CORPORATE SOURCE: Depto. Genetica, Fac. Med. de Ribeirao Preto-USP, Av.

Bandeirantes 3900, 14049.900 Ribeirao Preto, SP,

Brazil

SOURCE: Mutation Research, Genetic Toxicology (1996), 370(2),

121-126

CODEN: MGTOEB

PUBLISHER: Elsevier B.V.

DOCUMENT TYPE: Journal LANGUAGE: English

The radiotherapy treatment of human cancer is often limited by the side AΒ effects and complications induced in normal surrounding tissues. The use of therapeutic strategies that could protect normal tissues while permitting the death of malignant neoplasm would be advantageous. Some studies have suggested that the amino acid glutamine (GLN) can serve as a conditionally essential nutrient in patients in a catabolic condition. The objective of this study was to evaluate the possible radioprotection of GLN on the frequency of chromosomal aberrations, number of metaphases with chromosomal aberrations and mitotic index in bone marrow cells of Rattus norvegicus. In this in vivo test system, GLN was administered by gavage at concns. of 300 and 600 mg/kg body weight, in acute treatments, 30 min or 24 h before exposure to 3 Gy of whole-body gamma radiation. The results obtained in these expts. showed that GLN did not alter significantly the frequency of chromosome aberrations induced by gamma radiation under the exptl. conditions used in the present study.

L4 ANSWER 5 OF 5 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 1984:549106 CAPLUS

DOCUMENT NUMBER: 101:149106

ORIGINAL REFERENCE NO.: 101:22553a,22556a

TITLE: NMR study of in vivo RIF-1 tumors. Analysis of

perchloric acid extracts and identification of proton,

phosphorus-31, and carbon-13 resonances

AUTHOR(S): Evanochko, William T.; Sakai, Ted T.; Ng, Thian C.;

Krishna, N. Rama; Kim, Hyun Dju; Zeidler, Robert B.; Ghanta, Vithal K.; Brockman, R. Wallace; Schiffer,

Lewis M.; et al.

CORPORATE SOURCE: Univ. Stn., Comp. Cancer Cent., Birmingham, AL, 35294,

USA

SOURCE: Biochimica et Biophysica Acta, Molecular Cell Research

(1984), 805(1), 104-16

CODEN: BBAMCO; ISSN: 0167-4889

DOCUMENT TYPE: Journal LANGUAGE: English

Perchloric acid exts. of radiation-induced fibrosarcoma (RIF-1) AB tumors grown in mice have been analyzed by multinuclear NMR spectroscopy and by various chromatog. methods. This anal. has permitted the unambiguous assignment of the 31P resonances observed in vivo to specific phosphorus-containing metabolites. The region of the in vivo spectra generally assigned to sugar phosphates has been found in RIF-1 tumors to contain primarily phosphorylethanolamines and phosphorylcholine rather than glycolytic intermediates. Phosphocreatine was observed in exts. of these tumor cells grown in culture as well as in the vivo spectra, indicating that at least some of the phosphocreatine observed in vivo arises from the tumor itself and not from normal tissues. In the 31P-NMR spectra of the perchloric acid extract, resonances originating from purine and pyrimidine nucleoside di- and triphosphate were resolved. HPLC analyses of the nucleotide pool indicate that adenine derivs. were the most abundant components, but other nucleotides were present in significant amts. The 1H and 13C resonance assignments of the majority of metabolites present in RIF-1 exts. have also been made. Of particular importance is the ability to observe lactate, the levels of which may provide a noninvasive measure of glycolysis in these cells in both the in vivo and in vitro states. In addition, the aminosulfonic acid, taurine, was found in high levels in the tumor exts.

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FILE 'REGISTRY' ENTERED AT 14:38:03 ON 25 SEP 2009 L1 2 S GLUTAMINE/CN

FILE 'CAPLUS' ENTERED AT 14:38:19 ON 25 SEP 2009

L2 30059 S L1

L3 278 S L2 AND RADIATION

L4 5 S L3 AND NORMAL(A)TISSUE

=> s 13 and breast(A)cancer

100487 BREAST

822 BREASTS

100733 BREAST

(BREAST OR BREASTS)

422948 CANCER

62173 CANCERS 438285 CANCER

(CANCER OR CANCERS)

64858 BREAST(A)CANCER

L5 5 L3 AND BREAST(A)CANCER

=> d 15 1-5 ibib abs

L5 ANSWER 1 OF 5 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2008:1015298 CAPLUS

DOCUMENT NUMBER: 150:120585

TITLE: Glutamine affects glutathione recycling enzymes in a

DMBA-induced breast cancer model

AUTHOR(S): Kaufmann, Yihong; Todorova, Valentina K.; Luo, Shaoke;

Klimberg, V. Suzanne

CORPORATE SOURCE: Medical Research Service, Central Arkansas Veterans

Healthcare System, Little Rock, AR, USA
Nutrition and Cancor (2008) 60(4) 518-53

SOURCE: Nutrition and Cancer (2008), 60(4), 518-525

CODEN: NUCADQ; ISSN: 0163-5581

PUBLISHER: Taylor & Francis, Inc.

DOCUMENT TYPE: Journal LANGUAGE: English

Malignancy depletes host glutathione (GSH) levels to increase treatment-related toxicity and increases itself to resist the treatments. Our previous studies have shown that dietary glutamine (GLN) prevented 7,12-dimethylbenz[a]anthracene (DMBA)-induced mammary tumors through enhancing gut GSH release and reducing tumor GSH level. In addition, GSH synthesis, metabolism, and recycling are accomplished in  $\gamma$ -glutamyl cycle. We hypothesized that the GLN prevention might be through a differential regulation of the  $\gamma$ -glutamyl cycle enzymes. Female Sprague-Dawley rats were randomized into DMBA-tumor bearing, DMBA-treated, and control groups subdivided into GLN and water groups. GLN supplementation was given at 1 g/kg/day by gastric gavage. The activities and mRNA levels of  $\gamma$ -glutamyl transpeptidase (GTP),  $\gamma$ -glutamylcysteine synthetase (GCS), 5-oxo-L-prolinase (OPase),  $\gamma$ -glutamyl transferase (GTF), and glutaminase (GLNase) were determined in gut mucosa and breast tumor using specific enzyme assays and semiguant. reverse transcription polymerase chain reaction. GLN upregulated gut GTP, GCS, OPase, and GLNase in DMBA-tumor bearing, DMBA-treated, and/or control rats; however, it downregulated these enzymes in the tumor. The paradoxical effect of GLN on key GSH recycling enzymes in the gut vs. tumor suggests that dietary supplemental GLN could be used in the clin. practice to increase the therapeutic index of cancer treatments by protecting normal tissues from, and sensitizing tumor cells to, chemotherapy and radiation-related injury.

OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD

(2 CITINGS)

REFERENCE COUNT: 49 THERE ARE 49 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 2 OF 5 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2006:997702 CAPLUS

DOCUMENT NUMBER: 146:378886

TITLE: Modulation of p53 and c-myc in DMBA-induced mammary

tumors by oral glutamine

AUTHOR(S): Todorova, Valentina K.; Kaufmann, Yihong; Luo, Shaoke;

Klimberg, V. Suzanne

CORPORATE SOURCE: Division of Breast Surgical Oncology, Department of

Surgery, University of Arkansas for Medical Sciences,

Little Rock, AR, 72205, USA

SOURCE: Nutrition and Cancer (2006), 54(2), 263-273

CODEN: NUCADQ; ISSN: 0163-5581

PUBLISHER: Lawrence Erlbaum Associates, Inc.

DOCUMENT TYPE: Journal LANGUAGE: English

Previous studies established that oral glutamine (GLN) reduced tumor development in implantable and 7,12-dimethylbenz(a)anthracene (DMBA)-induced breast cancer models. This finding was associated with a decrease in tumor glutathione (GSH) levels, while maintaining normal gut, blood, and breast GSH. Alterations in GSH levels contribute to the control of apoptotic and cell cycle-regulating signaling. The aim of this study was to examine the role of dietary GLN on activation of p53 and c-myc, which play critical roles in cancer development and sensitivity to radiation and chemotherapy. Mammary gland carcinomas were induced in rats by DMBA. The rats were gavaged daily with GLN or water (controls), starting 1 wk prior DMBA-application and throughout the duration of the experiment (11 wk after DMBA). Tumor DNA was examined for mutations in p53 exons 5 and 6. Protein and mRNA levels of p53, p21WAF1/CIP1, PTEN, IGF-IR, mdm2, and c-myc in tumors of GLN-supplemented rats were compared with those of the control rats (received water). The sequencing of p53 showed that it was wild type. Increased phosphorylation of p53, as well as higher mRNA and protein levels of p21WAF1/CIP1, PTEN, and mdm2, and lower levels of IGF-IR were detected in tumors of GLN-supplemented rats vs. controls. Both phosphorylated c-myc and c-myc mRNA levels were reduced by GLN. The up-regulation of tumor p53 signaling and down-regulation of c-myc, in addition to previously established inhibition of Akt signaling in DMBAbreast cancer model, suggest that dietary GLN could be a

useful approach for increasing the effectiveness of cancer treatment. OS.CITING REF COUNT: 3 THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD

(3 CITINGS)

REFERENCE COUNT: 65 THERE ARE 65 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 3 OF 5 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2006:210688 CAPLUS

DOCUMENT NUMBER: 145:179905

TITLE: New strategies for management of oral mucositis in

cancer patients

AUTHOR(S): Peterson, Douglas E.

CORPORATE SOURCE: Head & Neck/Oral Oncology Program, Neag Comprehensive

Cancer Center, University of Connecticut Health

Center, Farmington, USA

SOURCE: Journal of Supportive Oncology (2006), 4(2, Suppl. 1),

9-13

CODEN: JSOOBY; ISSN: 1544-6794

PUBLISHER: Elsevier Inc.

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

A review. Oral mucositis can be a significant problem for cancer patients AB and is frequently seen in the patient population receiving high-dose head and neck radiation therapy (85%-100%), stem cell transplantation (75%-100%), and myelosuppressive chemotherapy for solid tumors (5%-40%). Current guidelines published through the joint efforts of the Multinational Association of Supportive Care in Cancer and the International Society for Oral Oncol. recommend strategies for the prevention and treatment of mucositis in the setting of radiation therapy, chemotherapy, and combined chemoradiation therapy. An improved understanding of its pathol. basis has led to the development of targeted agents to combat mucositis. One of these drugs, palifermin, is a keratinocyte growth factor agent approved for patients with hematol. malignancies receiving myelotoxic therapy requiring hematopoietic stem cell support. Another agent is AES-14, an uptake-enhanced L-glutamine suspension that has shown efficacy in phase III trials in reducing the

risk of developing oral mucositis in breast cancer patients receiving chemotherapy. As the understanding of the pathobiol. of mucositis improves, clinicians should be able to customize future therapies based on each patients risk for developing the condition.

OS.CITING REF COUNT: THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD 3

(3 CITINGS)

REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

ANSWER 4 OF 5 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2005:121209 CAPLUS

DOCUMENT NUMBER: 142:191334

TITLE: Compositions and methods for monitoring the use of glutamine supplementation in protecting breast tissue

against radiation injury during cancer

treatment

Suva, Larry J.; Klimberg, V. Suzanne INVENTOR(S):

PATENT ASSIGNEE(S): Aesgen, Inc., USA PCT Int. Appl., 24 pp. SOURCE:

CODEN: PIXXD2

DOCUMENT TYPE: Patent LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

P	PATENT NO.				KIND DATE			APPLICATION NO.						DATE				
W(	WO 2005012904			A1 20050210		WO 2004-US24928				20040730								
	W:	ΑE,	AG,	AL,	AM,	ΑT,	ΑU,	ΑZ,	ΒA,	BB,	BG,	BR,	BW,	BY,	BZ,	CA,	CH,	
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US	US 20050042700				A1 20050224			US 2004-903500					20040730					
US	US 7186517				B2 20070306													
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The present invention provides a method for monitoring the effectiveness AB of glutamine supplementation to protect breast tissue against radiation injury, the method comprising monitoring the concentration of a 9.29 kDa in serum of a human before, during, and after the administration of glutamine.

REFERENCE COUNT: THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

ANSWER 5 OF 5 CAPLUS COPYRIGHT 2009 ACS on STN

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DOCUMENT NUMBER: 135:251535

TITLE: Comparative evaluation of blood plasma and tumor

tissue amino acid pool in radiation or

neoadjuvant preoperative therapies of breast

cancer with the antitumor drug Ukrain

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AΒ This study comparatively evaluated free amino acid pool formation in patients with T1-3NO-2MO breast cancer treated with the drug Ukrain (25 patients, i.v. 100 mg/course) in combination with preoperative radiation or neoadjuvant therapies (25 subjects, total dose 20 Gy). All the patients underwent radical mastectomy. Preoperative radiation did not essentially change the range of the blood plasma parameters studied. However, the authors observed decreased concns. of blood plasma ornithine and citrulline and a reduced content of aminobutyric acid, as compared with levels on admission, which may indicate an acceleration of detoxication processes in the liver. comparison with healthy mammary gland tissue, the tumor tissue of the patients subjected to radiation therapy showed 1.5- to twofold increased concns. of cysteate, taurine, aspartate, glutamate, proline, glycine, alanine, valine, tyrosine and histidine, which substantiates the idea of tumor tissue being a trap for numerous energy and plastic substrates and indicates active transport of the above compds. into the tumor. The application of Ukrain had virtually no influence on concns. of the majority of blood plasma amino acids and derivs.: the total concentration

the compds. studied as well as the essential and nonessential amino acid pools remained unchanged. As compared with healthy breast tissue, the considerably increased levels of thiol-containing amino acids, such as methionine, cystine, cysteate and taurine, in the tumor tissue of patients receiving neoadjuvant therapy with Ukrain, indicates high activity of trans-sulfuration processes in this tissue. Simultaneously, in contrast to radiation therapy, Ukrain induced a marked dose-dependent increase in the concentration of proline in breast tumor tissue. The above changes were consistent with the results of the morphol. study which confirmed the emergence of numerous foci of necrosis in the tumor and indicated activation of Ukrain-induced proteolytic and degradation processes in the tumor. The results obtained have led the authors to conclude that a mechanism of Ukrain's cancerostatic effect is to control the transport and reactions of intermediate amino acid metabolism as well as to activate proline biosynthesis in the tumor, causing enhanced development of connective tissue. It is suggested that an important practical conclusion from the present study is the lack of damaging effect of preoperative radiation therapy in the above regimen and the favorable (normalizing) action of Ukrain, at a course dose of 100 mg, on the amino acid pool formation in the organism of patients with breast cancer.

OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD

(1 CITINGS)

REFERENCE COUNT: 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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(FILE 'HOME' ENTERED AT 14:37:49 ON 25 SEP 2009)

FILE 'REGISTRY' ENTERED AT 14:38:03 ON 25 SEP 2009 L1 2 S GLUTAMINE/CN

FILE 'CAPLUS' ENTERED AT 14:38:19 ON 25 SEP 2009

L2 L3 L4 L5	5	S S	L2 L3	AND	RADIATION NORMAL(A)TISSUE BREAST(A)CANCER
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	ENTRY	SESSION
FULL ESTIMATED COST	45.70	53.80
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